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Critical Illness-Related Corticosteroid Insufficiency (CIRCI)

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Table 1. Main Mechanisms of Critical Illness Related Corticosteroid Insufficiency

General defect	Main mechanisms	Key factors
Decrease in cortisol production		
Altered adrenal synthesis of cortisol	Necrosis/hemorrhage	Acute kidney failure; hypo-coagulation; disseminated intravascular coagulation; cardiovascular collapse; tyrosine kinase inhibitors
	Decreased availability of esterified cholesterol	Depletion in adrenal storage regulated by annexin A1 – formyl peptide receptors Down-regulated Scavenger Receptor-B1
	Inhibition of steroidogenesis	Immune cells / Toll like receptors /cytokines
		Drugs (e.g., sedatives, corticosteroids
		ACTH-like molecules (e.g., corticostatins
Altered synthesis of CRH/ACTH	Necrosis/hemorrhage	Cardiovascular collapse; disseminated intravascular coagulation; treatment with vasopressor agents
	Inhibition of ACTH synthesis	Glial cells / nitric oxide mediated neuronal apoptosis
		Increased negative feedback from circulating cortisol following up-regulation of ACTH-independent mechanisms of cortisol synthesis
		Drugs (e.g., sedatives, anti-infective, psychoactive agents
		Inappropriate cessation of glucocorticoid treatment
Alteration of cortisol metabolism	Decreased cortisol transport	Down-regulation of liver synthesis of cortisol-binding globulins and albumin
	Reduced cortisol breakdown	Decreased expression and activity of the glucocorticoid inactivating 5-reductase enzymes in the liver with putative role of bile acids; Decreased expression and activity of the hydroxysteroid dehydrogenase in the kidney
Target tissue resistance to cortisol	Inadequate glucocorticoid receptor alpha (GR-α) activity	Decreased expression and decreased transcription; unclear mechanisms – NF-kappa B driven?